

Importance of Caloric Intake During Renal Failure

FRANK H. CARTER, M.D., and ROBERT T. PLUMB, M.D., San Diego

IN ACUTE RENAL FAILURE the end-products of protein metabolism are retained—creatinine, uric acid, phosphorus and sulfates among them. These cause metabolic acidosis and contribute to uremic sequelae in the lungs, gastrointestinal tract and pericardium, as well as in the vascular and nervous systems. When fat and carbohydrate intake is inadequate, protein becomes the main source of calories, and protein metabolic wastes accumulate rapidly. It has been estimated that 20 to 30 grams of protein intake is sufficient to maintain nitrogen balance;⁴ any additional protein is used for energy and further wastes accumulate. Moreover, stress, infection, fever or other hypermetabolic states mobilize body protein in more than normal amounts, and the increased wastage from this cause is compounded by the resulting mobilization of potassium, a further toxic load. Some replacement of essential amino acids metabolized by stress and by tissue repair may be necessary, but as yet this requirement has not been assessed.

By providing readily available fat or carbohydrate, protein is spared as a caloric source and the accumulation of retained protein waste is retarded. Energy is provided to drive potassium into the cells and the danger of hyperkalemia is diminished. During renal failure fat is not completely oxidized; keto acids and acetate accumulate, and the keto acids contribute to metabolic acidosis. (Normally, these substances enter the carbohydrate cycle and are converted into carbon dioxide and water.) Recent studies² indicate that stored body fat constitutes a considerable part of the caloric supply. If accumulation of keto acids is to be reduced, adequate carbohydrate must be provided for the complete oxidation of fat, for depression of protein oxidation with its release of indisposable wastes, and for the return of potassium to the cells.

The feeding of carbohydrates to a patient in renal failure may present a serious problem. As a first consideration, it must be remembered that the end-products of carbohydrate metabolism are carbon dioxide and water. The water, then, will contribute to overhydration; hence it must be considered in the daily computation of fluid replacement. Second, there is the clinical problem of encouraging

• Loss of excretory function in acute renal failure results in the retention of catabolites and fluid. In the absence of available carbohydrate, endogenous fat and protein become the main caloric sources. This results in the rapid accumulation of keto acids and nitrogenous wastes. By providing readily available non-nitrogenous calories, protein catabolism is reduced, complete oxidation of fat is obtained and energy is provided to drive potassium into the intracellular compartment.

The patient should be encouraged to eat despite his apathy, fear of vomiting and characteristically paranoid mood. Tube or parenteral feeding is complicated by the need to restrict fluid. Numerous small feedings are more successful than large meals. Hard candy and alcohol are often acceptable sources of calories, fat emulsions seldom. Oral hygiene aids feeding, and tranquilizers and anticholinergics are useful.

If the patient does not take food by mouth, tube feeding may be carried out. Because of the bleeding tendency so often occurring in uremia, tube feeding may be contraindicated if it causes erosion of the pharynx or esophagus. Intravenous infusion of invert sugar, glucose and alcohol may be necessary if nutrition cannot be accomplished by other means.

carbohydrate intake in a patient tending toward nausea, vomiting, apathy, and—another frequent feature of uremic intoxication—paranoid reaction. There is a basic disinclination to eat, and fasting presents as many difficulties as protein feeding, since in both conditions body protein becomes the main caloric source. The necessity to limit fluid further complicates administration of calories.

METHODS OF TREATMENT

During the early phase of renal shutdown, caloric requirements can often be met by diet alone. The patient must be informed of the importance of adequate intake, and every effort should be made to encourage the waning appetite. Since fear of vomiting becomes an obsession with many patients, it should be explained that after initial emesis, additional food taken may be retained.

Dietary planning should include: Intake of 2,000 to 2,400 calories daily; limitation of protein to less than 50 gm. daily; sodium limitation to 500 mg. daily (unless abnormal losses occur); severe limitation of potassium-containing foods.

Five or six small feedings are generally better tolerated than three large meals. An estimate of the

From the Department of Urology, San Diego County General Hospital, San Diego.

Presented before the Section on Urology at the 87th Annual Session of the California Medical Association, Los Angeles, April 27 to 30, 1958.

actual intake should be made at the time of each meal, so that compensatory calories may be administered parenterally.

Caloric supplements to the diet should be tried. Many patients can retain additional carbohydrate in the form of hard candy or plain sugar. Starvation ketosis can be controlled by giving 100 gm. of carbohydrate which should be considered the minimum daily replacement. Ingestible fat emulsions provide four to five calories per milliliter and are satisfactory for some patients, but in many they cause nausea, vomiting, and diarrhea. Alcoholic beverages used in moderation boost the caloric intake and provide a mild sense of well-being. The Borst regimen⁶ of flour soup and butter balls may be tried, but it is unpalatable and generally refused by patients.

Oral dryness and encrustation make feeding difficult. Careful attention to care of the mouth helps to obviate these problems.

As retention products accumulate, anorexia increases. Chlorpromazine given 30 to 60 minutes before meals helps to overcome nausea. Urecholine, 5 to 10 mg. every six hours, or prostigmine (1:4000), 1 ml. at six-hour intervals, often improves peristalsis.

With progression of uremia, oral feeding becomes extremely difficult, but as long as peristalsis continues, fluid and caloric requirements may be supplied through a small plastic nasogastric tube. In most hospitals, the diet can be blended, strained and diluted to provide a tube feeding suitable for slow continuous drip administration. Blended foods seem to be tolerated better than fat or carbohydrate concentrates. A satisfactory pump has been devised¹ to force thicker fluids through plastic tubes at a slow continuous rate. This adjunct may help to increase caloric intake. Repeated observation for erosion in the nasopharynx is necessary, as severe bleeding may ensue. The bleeding diathesis that often occurs in uremia contraindicates the use of a nasogastric tube. Tube feeding should be discontinued if the abdomen becomes silent.

Further progression into the uremic state produces intestinal atonia and complete revulsion toward alimentary intake. At this point, it is necessary to resort to continuous parenteral nutrition. A cannula placed in the superior vena cava (via external jugular or cephalic vein), or in the inferior vena cava (via superficial external pudendal or saphenous vein) can be used for both hypertonic parenteral fluid administration and dialysis. Such a cannula should remain effective for two to three weeks with

proper care. Heparin sodium, 50 mg. added to each liter of fluid, usually maintains the patency of the cannula. If the cannula is inserted through a stab wound, better wound healing can be obtained and infection is diminished. This procedure allows the initial incision to heal per primum.³

The fluids of choice are 40 per cent invert sugar and 50 per cent glucose. Regular insulin, one unit for each 5 gm. of sugar, should be added to each infusion. One hundred milliliters of 95 per cent ethyl alcohol, used as a supplement, provides about 400 calories. When diluted in one liter of fluid, this provides a sense of well-being without danger of intoxication. As yet, fat emulsions satisfactory for intravenous administration are not readily available. The ideal solution would be a combined fat and carbohydrate mixture of stable composition, free of pyrogenic ingredients.

To insure slow even infusion over any 24-hour period, infusion bottles should be marked at hourly-level intervals and the consumption checked frequently by attendants.

Severe vitamin depletion is a concomitant of the uremic state. Vitamin replacement carried out as nutrients are being given enhances utilization of caloric intake.

No single method of treatment is ideal. Each patient must be critically observed through each stage in the progression of the uremic process. Each mode of therapy must be analyzed and must be discarded when it no longer serves the intended purpose. The wide range of patient response necessitates a varied armamentarium.

2001 Fourth Avenue, San Diego 1 (Carter).

REFERENCES

1. Barron, J., Prendergast, J. J., and Jocz, M. W.: Food pump; new approach to tube feeding, *J.A.M.A.*, 161:621-622, June 16, 1956.
2. Bluemle, L. W., Jr., Potter, H. P., and Elkinton, J. R.: Changes in body composition in acute renal failure; *J. Clin. Invest.*, 35:1094-1108, Oct. 1956.
3. Carter, F. H., Aoyama, S., Mercer, R. D., and Kolff, W. J.: Hemodialysis in children, *J. Peds.*, 51:125-136, Aug. 1957.
4. Kempner, W.: Compensation of renal metabolic dysfunction; treatment of kidney disease and hypertensive vascular disease with rice diet, *N. Carolina Med. J.*, 6:61, Feb. 1945.
5. Kolff, W. J.: Forced high caloric low protein diet in the treatment of uremia, *Ann. N. Y. Acad. Sci.*, 56:107-121, Oct. 1952.
6. Kolff, W. J.: Acute renal failure; causes and treatment, *Med. Clin. N. Amer.*, 39:1041-1071, July 1955.
7. Merrill, J. P.: The treatment of renal failure, New York, N. Y. Grune and Stratton, Publishers, 1955.